Carbon disulfide

75-15-0

$S = C = S$

I. Physical and Chemical Properties

Description	Clear, colorless or faintly yellow liquid
Molecular formula	CS_2
Molecular weight	76.14
Air concentration conversion	3.1 mg/m^3 per ppm at 25^oC

II. Overview

Neurotoxicity is one of the key toxicological endpoints of concern for infants and children. A primary target of carbon disulfide (CS_2) toxicity is the nervous system. The major neurotoxic actions of CS_2 measured in occupationally-exposed adults are the acute development of mental disturbances and the chronic development of neurophysiological and neuropathology changes (decreased peripheral nerve impulse conduction, motor and/or sensory neuropathies, cerebral or cerebellar atrophy, and neuropsychological organic changes) (Aaserud *et al.* 1988, 1990, 1992; Foa *et al.*, 1976; Hirata *et al.* 1992; Ruijten *et al.* 1990, 1993). Neuropathy and cardiovascular effects occur with chronic occupational exposures of 10 to 20 mg/m^3 (Johnson *et al.*, 1983; Vanhoorne et al., 1992; 1995). Animal studies have also shown that CS_2 is a neurotoxicant, primarily indicated by pathological changes in the nervous system. Transient delays in behavioral development have been reported in young animals exposed to as low as 10 mg/m^3 (3 ppm) CS_2 (Tabacova and Balabaeva, 1980).

There is some evidence of increased sensitivity to acutely lethal CS_2 exposures and lower detoxification rates of CS_2 in newborn animals.

There is some evidence of teratogenic and delayed developmental effects following exposure to CS_2 in animal studies. Developmental toxicity is another key toxicological endpoint for infants and children. Damage sustained during *in utero* exposure poses a risk of adverse postnatal health effects and, as such, it is necessary to consider developmental toxicity following prenatal exposure. The teratogenic findings among available studies do not present a consistent pattern of specific effects.

III. Principal Sources of Exposure

The most prominent industrial use of and source of occupational exposures to CS_2 has been in the production of viscose rayon fibers. There are no such facilities in California. Other uses of CS_2 include in the production of carbon tetrachloride and cellophane, and as a solvent for rubber, sulfur, oils, resins and waxes. In the past, CS_2 was used in soil fumigation and insect control in stored grain. Industrial processes that produce CS_2 as a by-product include coal blast furnaces and oil refining (HSDB, 1995). The annual statewide emissions from facilities reporting under the Air Toxics Hot Spots Program in California based on the most recent inventory were estimated to be 1561 pounds of CS_2 (CARB, 2000), largely as fugitive emissions from refineries. CS_2 is not routinely monitored in ambient air in California.

IV. Potential for Differential Effects

A. Summary of Key Human Studies

There are no human studies that directly address age-related susceptibility. While occupationally exposed workers have been examined in several studies, there are no studies of children exposed to CS_2.

In an abstract of an epidemiological study of birth defects among female workers occupationally exposed to CS_2, Bao et al. (1991) reported an increased rate of birth defects (2.6% vs. 1.3%) among 682 exposed women compared to 745 women in the control group. The most common defects were congenital heart defects, inguinal hernia, and CNS defects. However, there was no significant difference in birth defects between those with estimated exposures greater than 10 mg/m^3 compared to those with lower exposures. There were no differences in rates of stillbirth, low birth weight, or neonatal or perinatal deaths among any of the groups.

A primary target of CS_2 toxicity is the nervous system. Neurotoxicity is one of the toxicological endpoints of concern for infants and children. The major neurotoxic action of CS_2 measured in adults in occupational settings is the development of mental disturbances, such as change of personality, irritability, and forgetfulness. These are often accompanied by neurophysiological and neuropathological changes after prolonged exposure; decreased peripheral nerve impulse conduction, motor and/or sensory neuropathies, cerebral or cerebellar atrophy, and neuropsychological organic changes (Aaserud *et al*. 1988, 1990, 1992; Foa *et al.*, 1976; Hirata *et al*. 1992; Ruijten *et al*. 1990, 1993). Alterations in behavioral indices measured in adults have been historically associated with high levels of CS_2, often in the excess of 20 ppm (Foa *et al.* 1976; Hannien *et al.*, 1978).

Studies have identified alterations in the nerve conduction of workers chronically exposed to lower CS_2 levels (Hirata *et al.*, 1992; Johnson *et al.*, 1983; Ruijten *et al.*, 1990, 1993). A cross-sectional study of Japanese spinning workers identified alterations in the central nervous system as measured by brain stem auditory evoked potential (BAEP) (Hirata *et al.*, 1992). The latencies of the three main BAEP components increased significantly in the CS_2 exposed workers (more than 20 years duration) when compared to controls. CS_2 exposures ranged from 3.3 to 8.2 ppm (mean 4.76 ppm). Ruijten *et al.* (1993) identified mild presymptomatic nerve impairment (decreased conduction velocities and response amplitudes) in 44 CS_2-exposed workers with an average cumulative exposure ranging from 192 to 213 ppm-year (mean duration 26.1 years).

In an occupational study evaluating the effects of CS_2 exposure on the peripheral nervous system, Johnson *et al.* (1983) identified a significant dose related reduction in the motor nerve conduction velocities in the calves and ankles of workers exposed to high (median 7.6 ppm) CS_2 levels versus a comparison group (median 0.2 ppm). Since the motor nerve conduction velocity, although reduced, was still within normal values, the authors considered the measured difference an indication of minimal neurotoxicity. The mean exposure concentration for all exposed workers (n = 145) ranged from 0.6 to 16 ppm (mean 7.3 ppm) with a mean 12.1 year duration. This study established a chronic LOAEL of 7.6 ppm for minor neurological effects (decreased peroneal nerve conduction velocity and sural nerve conduction velocity).

A second epidemiological study of interest has been partially reported by Vanhoorne and colleagues (1995). A group of 111 Belgian viscose rayon factory workers were exposed to 4 to 112 mg/m^3 CS_2 (time-weighted average 1 to 40 mg/m^3). Among four categories of cumulative exposure (0, 1 to 300, 301 to 600, and greater than 600 mg/m^3 years), a clear dose-response effect was observed for reduced mean peroneal motor nerve conduction velocities in both fast and slow fibers. Unfortunately, the data are incompletely reported, and the mean duration of exposure is not given. The lowest exposure group (1 to 300 mg/m^3 year; 0.36 to 11 mg/m^3 year TWA) may be associated with a significant reduction (approximately 5 to 10%) in peroneal motor nerve conduction velocity. Assuming the exposure duration was similar to that of the Johnson study (12.1 years), the equivalent TWA concentrations associated with 300 or 600 mg/m^3 year exposure are 2.8 and 5.6 ppm, respectively.

B. Summary of Key Animal Studies

The 24-hr lethal ip LD_{50} values for CS_2 were estimated in 1-, 5-, 10-, 20-, 30- and 40-day-old rats (sample size not specified) (Green and Hunter, 1985). 1-day-old rats (LD_{50} 583 mg/kg, ip) were about 3-times more susceptible than 20-day-old rats (LD_{50} 1545 mg/kg, ip).

^{14}C- and ^{35}S-labelled CS_2 was given ip to 1-, 5-, 10-, 20-, 30-, and 40-day-old rats (Snyderwine and Hunter, 1987). Thirty- and forty-day-old rats (sample size not reported) metabolized significantly more CS_2 to CO_2 and expired significantly less CS_2 than 1- to 20-day-old rats. Twenty-four hr after administration, up to 13 times more ^{35}S -label (radioactivity per g of tissue) were present in organs from 1-day-old rats than in similar organs from 40-day-old rats. The study does not specifically address the toxicological implications of the metabolic differences, and did not include fully mature animals. However, inability to detoxify CS_2 would lead to higher tissue concentrations and thus, potentially, increased toxicity.

New Zealand white rabbits (24 per group) inhaled 0, 60, 100, 300, 600 or 1200 ppm CS_2 for 6 h/d on gestation days 6 to 18 (Pathology Associates, 1991). Developmental toxicity (NOAEL = 300 ppm; 930 mg/m^3) was noted at concentrations lower than those associated with significant maternal toxicity (NOAEL = 600 ppm; 1860 mg/m^3) (Pathology Associates, 1991). The adults did have some slight hematological changes at the 600 ppm level, but the authors questioned the biological significance of these marginal findings. Reduced fetal body weights were noted at 600 and 1200 ppm. Cumulative malformations were increased in the 1200 (3720 mg/m^3) but not 600 ppm group, though there were no significant increases in any specific malformation in any group. Maternal effects at 1200 ppm were decreased body weight, ataxia, wheezing, and tremors. In an initial range-finding study, exposure to 3000 ppm was associated with significant lethality.

Rats were exposed to 100 mg/m^3 (32 ppm) for 4 hr/d on gestation days 7 and 8, and the embryos explanted to culture medium at day 9.5. Growth of explants of 10 treated and 17 control embryos was monitored for 44 hours. CS_2 at this concentration induced growth retardation in treated embryos relative to controls (Zhao et al., 1997).

In a two-generation study, Tabacova et al. (1983) exposed pregnant Albino rats (30-32 pregnant females per group) to CS_2 (0.03, 10, 100, or 200 mg/m^3). The two highest dose levels were both teratogenic and maternally neurotoxic. There were no significant adverse effects in the F1 generation at the 2 low dose levels. However, significant increases in teratogenicity were found in the F2 generation at 10 mg/m^3, as well as increased postnatal neurological effects including hypoactivity, mild ataxia and gait disturbances, hind-limb weakness, spinning and tremor (Tabacova et al., 1983). While the overall rate of malformations (club foot, hydrocephalus, microcephalus, generalized edema) exhibited a dose-response trend, with increased effects in the F2 generation, the specific malformations exhibited a less-consistent pattern. For example, while club foot was the predominant malformation in the F1 fetuses (occurring at 100 and 200 mg/m^3); much lower rates of club foot were noted in the F2 generation (including none in the 200 mg/m^3 group). Limitations of the study include a lack of information on chemical purity and exposure methods, lack of concurrent controls, lack of clear dose-response trend, and incomplete reporting on the statistical significance of reported behavioral effects.

Wistar albino rats (32 animals per group) were exposed to 50, 100, or 200 mg/m^3 CS_2 for 8 hours per days throughout gestation. There were no statistically significant results in the 50 mg/m^3 group. In the 100 and 200 mg/m^3 groups, there were statistically significant increases in reduced fetal body weights, and reduced post natal body weights for 21 days, which subsequently disappeared. There was an increase in external malformations (hydrocephalus, club foot, and tail deformations) at the two higher doses (Tabacova et al., 1978).

Behavioral effects were examined in the offspring of Lati:CFY rats (8 per group) exposed to CS_2 (0, 10, 700, or 2000 mg/m^3) for 6 hours per days over days 7 to 15 of gestation. The two high doses caused significant perinatal mortality. Avoidance conditioning was tested using a bell as a conditional stimulus prior to an electric shock. The animals learned to avoid the shock by jumping onto a pole at the sound of the bell. The latency to jump onto the pole and errors were measured as a means to evaluate avoidance conditioning in the treated versus control animals. The authors reported that there was a dose-related change in avoidance conditioning among male pups over the first 15 days (Lehotsky et al., 1985). While the magnitude of the effect on avoidance conditioning was greater at all doses relative to controls, and at 2000 mg/m^3 compared with 700 mg/m^3, the effect was virtually identical between the 10 and 700 mg/m^3. This lack of dose-response effect raises some question about the significance of this finding.

Effects of low (0.03 and 10 mg/m^3) prenatal exposures (8 hours per day throughout gestation) of CS_2 were studied in Wistar albino rats. No congenital malformations or significant prenatal effects were found in the 9-11 litters evaluated at each dose. Mortality during postnatal days 10 through 21 was increased in the 10 mg/m^3 group. Delays in the development of visual and auditory function were reported in the higher dose group (Tabacova and Balabaeva, 1980). There was no mention of maternal toxicity in this study.

Several other studies yielded either no teratogenic effects or effects only at maternally toxic exposures. Saillenfait et al. (1989) exposed rats via inhalation to 0, 100, 200, 400, or 800 ppm CS_2 for 6h/d during days 6-20 of gestation. Lower exposures (100 or 200 ppm; 310 or 620 mg/m^3) were not associated with maternal toxicity or adverse effects on the developing embryo or fetus. Higher concentrations (400 or 800 ppm; 1240 or 2480 mg/m^3) yielded a significant reduction of maternal weight gain as well as reductions of fetal body weight and a low incidence of club foot. Significant increases in unossified sternebrae were reported following 800 ppm (2480 mg/m^3) exposures. Nemec et al. (1993) reported no teratogenicity or maternal, developmental, or reproductive toxicity among pregnant CD rats and their offspring following exposure to 125 or 250 ppm (388 or 775 mg/m^3) from 2 weeks prior to mating through gestation day 19. At 500 ppm, dams had decreased body weight gain and food consumption; decreased litter viability but no teratogenic effects were also noted. CS_2 was not found to be teratogenic or embryotoxic following intraperitoneal administration to rats on days 1-15 of gestation (Beliles et al., 1980; Hardin et al., 1981). No significant effects were noted in animal inhalation exposures (20 to 40 ppm; 62 to 125 mg/m^3 CS_2) with either rats on days 1-19 of gestation or rabbits on days 1-24 of gestation.

Animal studies have also shown that CS_2 is a neurotoxicant not only in the developmental studies but also in adult animals. The neuropathologic changes consistently observed in rodents following CS_2 exposure include axonal swelling, demyelination, swelling at neuromuscular junctions, muscle atrophy and degeneration, damage to terminal axons, and nerve fiber breakdown (Clerici and Fechter, 1991; Colombi *et al.* 1981; Eskin *et al.*, 1988; Jirmanova and Lukas, 1984; Maroni *et al.*, 1979; Szendzikowski *et al.*, 1973). These adverse effects have been observed over a range of doses (250 to 800 ppm; 775 to 2480 mg/m^3), but few studies have attempted to establish a dose response for this CS_2-induced neurotoxicity.

In a 90 day subchronic inhalation study, Sprague Dawley and Fisher 344 rats exposed discontinuously (6 hours/day, 5 days/week) to CS_2 developed morphological alterations in nerves including axonal swelling and myelin degradation (Gottfried *et al.*, 1985). This study established a subchronic NOAEL of 50 ppm (155 mg/m^3) and a LOAEL of 300 ppm (930 mg/m^3) for morphological changes in nerves. A longer inhalation study in Wistar rats observed impairment in the conduction velocity of the sciatic and tibial nerves after 6 and 12 months of intermittent exposure to 289 ppm CS_2 (LOAEL of 289 ppm, 895 mg/m^3) (Knobloch *et al.*, 1979).

V. Additional Information

A. Other Toxicity

Although cardiovascular toxicity has not been singled out as an effect of concern for infants and children, there is little information on impacts of such toxicants on children's health. It is worth noting that CS_2 is associated with significant cardiovascular disease in occupational settings. Vascular atherosclerotic changes are also considered a major effect of chronic CS_2 exposure. Several occupational studies have demonstrated an increase in the mortality from ischemic heart disease in CS_2 exposed workers (Hernberg *et al.*, 1970; MacMahon and Monson, 1988; Tiller *et al.*, 1968; Tolonen *et al.*, 1979). A 2.5-fold excess in mortality from coronary heart disease in workers exposed to CS_2 was first reported by Tiller *et al.* (1968). A subsequent prospective study by Hernberg *et al.* (1970) found a 5.6-fold increased risk in coronary heart disease mortality and a 3-fold increased risk of a first nonfatal myocardial infarction in CS_2 exposed workers.

Egeland *et al.* (1992) and Vanhoorne *et al.* (1992) have reported that human exposure to CS_2 for more than one year causes changes in diastolic blood pressure, low density lipoprotein cholesterol, and apolipoproteins A1 and B. Egeland *et al.* (1992) used cross sectional data on 165 CS_2-exposed workers (245 controls) collected in 1979 by Fajen *et al.* (1981). Workers were exposed for at least 1 year in a viscose rayon factory to an estimated median TWA (8-hour) of 7.6 ppm. The Egeland *et al.* (1992) study indicated that modest CS_2 exposure (range 3.4 to 5.1 ppm, median 4.1 ppm (12.7 mg/m^3)) was associated with increased low density lipoprotein cholesterol (LDLc), which has been associated with atherosclerotic heart disease. No significant differences were seen between controls and the low CS_2 exposed group (range 0.04 to 1.02 ppm, median 1.00 ppm). This study indicates a chronic NOAEL of 1.00 ppm (3.1 mg/m^3) and a LOAEL of 4.1 ppm (12.7 mg/m^3) for increased LDLc and diastolic blood pressure. Vanhoorne *et al.* (1992) identified increased LDL-cholesterol, apolipoprotein B, systolic and diastolic blood pressure indicative of an increased coronary risk in workers from a Belgium viscose rayon factory (115 exposed and 76 controls). CS_2 concentrations ranged from 1 to 36 ppm (3.1 to 112 mg/m^3). Duration of exposure was not indicated. Even though these biochemical changes were observed, no significant increases in mild cardiovascular disease, such as angina, myocardial infarction, or ischemia were determined by ECG changes.

Wronska-Nofer (1973) showed a positive relationship between the level of triglycerides, the rate of cholesterol synthesis, and CS_2 exposure in Wistar rats exposed to 0, 73.8, 160, 321 or 546 ppm CS_2 for 5 hours/day, 6 days/week over 8 months. This study found a subchronic LOAEL of 73.8 ppm (229 mg/m^3) for disturbances in lipid metabolism (increase in serum cholesterol and serum triglycerides).

B. Regulatory Background - Brief description of RELs and URF

OEHHA is currently developing a chronic REL for CS_2 that is tentatively based on benchmark concentration modeling of neuropathy following chronic occupational exposure to carbon disulfide. A US EPA reference concentration (RfC) based on benchmark concentration (BMC) modeling (10% response) of neurotoxicity in occupationally exposed workers reported by Johnson and colleagues

(1983) was 0.7 mg/m^3 (0.2 ppm) (U. S. EPA, 1995). ATSDR, also using a LOAEL of 7.6 ppm from the Johnson data developed a chronic-duration inhalation MRL of 0.3 ppm. Environment Canada, using a BMC for 5% response derived from Johnson study, derived a Tolerable Concentration (TC) for CS_2 of 0.1 mg/m^3 (0.03 ppm) (Environment Canada, 2000).

VI. Conclusions

Carbon disulfide is a neurotoxicant in humans and animals. Neurotoxicity is a key toxicological endpoint of concern for infants and children. There is some evidence of developmental toxicity in animals, although the picture is inconsistent in terms of a clear dose-response relationship. Developmental toxicity is a key toxicological endpoint for infants and children, and damage sustained from prenatal exposure poses adverse health impacts postnatally. Exposures in ambient air in California are generally quite low as there are few emissions sources. Thus, OEHHA placed carbon disulfide into Tier 2. Should information arise indicating that localized exposures are significant, OEHHA may revisit listing carbon disulfide under SB 25.

VII. References

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